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=> d all tot 144

L44 ANSWER 1 OF 7 MEDLINE on STN  
AN 2005246876 MEDLINE  
DN PubMed ID: 15887106  
TI Recombinant probiotics for treatment and prevention of **enterotoxigenic Escherichia coli** **diarrhea**.  
AU Paton Adrienne W; Jennings Michael P; Morona Renato; Wang Hui; Focareta Antonio; Roddam Louise F; Paton James C  
CS School of Molecular and Biomedical Science, University of Adelaide, South Australia, Australia.  
SO Gastroenterology, (2005 May) Vol. 128, No. 5, pp. 1219-28.  
Journal code: 0374630. ISSN: 0016-5085.  
CM Comment in: Gastroenterology. 2005 May;128(5):1509-12. PubMed ID: 15887131  
CY United States  
DT Journal; Article; (JOURNAL ARTICLE)  
(RESEARCH SUPPORT, NON-U.S. GOV'T)  
LA English  
FS Abridged Index Medicus Journals; Priority Journals  
EM 200507  
ED Entered STN: 12 May 2005  
Last Updated on STN: 6 Jul 2005  
Entered Medline: 5 Jul 2005  
AB BACKGROUND & AIMS: We have developed a therapeutic strategy for gastrointestinal infections that is based on molecular mimicry of host receptors for bacterial toxins on the surface of harmless gut bacteria. The aim of this study was to apply this to the development of a recombinant probiotic for treatment and prevention of **diarrheal** disease caused by **enterotoxigenic Escherichia coli** strains that produce heat-labile **enterotoxin**.  
METHODS: This was achieved by expressing glycosyltransferase genes from *Neisseria meningitidis* or *Campylobacter jejuni* in a harmless **Escherichia coli** strain (CWG308), resulting in the production of a chimeric lipopolysaccharide capable of binding heat-labile **enterotoxin** with high avidity. RESULTS: The strongest heat-labile **enterotoxin** binding was achieved with a construct (CWG308:pLNT) that expresses a mimic of **lacto-N-neotetraose**, which neutralized > or = 93.8% of the heat-labile **enterotoxin** activity in culture lysates of diverse **enterotoxigenic Escherichia coli** strains of both human and porcine origin. When tested with purified heat-labile **enterotoxin**, it was capable of adsorbing approximately 5% of its own weight of toxin. Weaker toxin neutralization was achieved with a construct that mimicked the ganglioside GM2. Preabsorption with, or coadministration of, CWG308:pLNT also resulted in significant *in vivo* protection from heat-labile **enterotoxin**-induced fluid secretion in rabbit ligated ileal loops. CONCLUSIONS: Toxin-binding probiotics such as those described here have considerable potential for prophylaxis and treatment of **enterotoxigenic Escherichia coli**-induced

travelers' diarrhea.

CT Adrenal Glands: CY, cytology

Animals

Bacterial Toxins: ME, metabolism

Campylobacter jejuni: GE, genetics

Cells, Cultured

Cholera Toxin: ME, metabolism

\*Diarrhea: MI, microbiology

\*Diarrhea: PC, prevention & control

Enterotoxins: ME, metabolism

\*Escherichia coli: CL, classification

\*Escherichia coli: GE, genetics

Escherichia coli: ME, metabolism

Glycosyltransferases: GE, genetics

Ileum: MI, microbiology

Lipopolysaccharides: ME, metabolism

Neisseria meningitidis: GE, genetics

\*Probiotics: PD, pharmacology

Rabbits

Recombinant Proteins: GE, genetics

RN 9012-63-9 (Cholera Toxin)

CN 0 (Bacterial Toxins); 0 (Enterotoxins); 0 (Lipopolysaccharides); 0 (Recombinant Proteins); 0 (heat stable toxin (*E coli*)); EC 2.4.- (Glycosyltransferases)

L44 ANSWER 2 OF 7 MEDLINE on STN

AN 2001060702 MEDLINE

DN PubMed ID: 10965049

TI Common architecture of the primary galactose binding sites of *Erythrina corallodendron* lectin and heat-labile **enterotoxin** from *Escherichia coli* in relation to the binding of branched **neolactohexaosylceramide**.

AU Teneberg S; Berntsson A; Angstrom J

CS Institute of Medical Biochemistry, Goteborg University, P.O. Box SE 405 30 Goteborg, Sweden.

SO Journal of biochemistry, (2000 Sep) Vol. 128, No. 3, pp. 481-91. Journal code: 0376600. ISSN: 0021-924X.

CY Japan

DT (COMPARATIVE STUDY)

Journal; Article; (JOURNAL ARTICLE)

(RESEARCH SUPPORT, NON-U.S. GOV'T)

LA English

FS Priority Journals

EM 200012

ED Entered STN: 22 Mar 2001

Last Updated on STN: 18 Dec 2002

Entered Medline: 22 Dec 2000

AB The heat-labile **enterotoxin** from *Escherichia coli* (LT) is responsible for so-called traveller's diarrhea and is closely related to the cholera toxin (CT). Toxin binding to GM1 at the epithelial cell surface of the small intestine initiates the subsequent diarrheal disease. However, LT has a broader receptor specificity than CT in that it also binds to N-acetyllactosamine-terminated structures. The unrelated lectin from *Erythrina corallodendron* (ECorL) shares this latter binding property. The findings that both ECorL and porcine LT (pLT) bind to lactose as well as to **neolactotetraosylceramide** suggests a common structural theme in their respective primary binding sites. Superimposing the terminal galactose of the lactoses in the respective crystal structures of pLT and ECorL reveals striking structural similarities around the galactose

despite the lack of sequence and folding homology, whereas the interactions of the penultimate GlcNAcb3 in the **neolactotetraosylceramide** differ. The binding of branched **neolactohexaosylceramide** to either protein reveals an enhanced affinity relative to **neolactotetraosylceramide**. The b3-linked branch is found to bind to the primary Gal binding pocket of both proteins, whereas the b6-linked branch outside this site provides additional interactions in accordance with the higher binding affinities found for this compound. While the remarkable architectural similarities of the primary galactose binding sites of pLT and ECORL point to a convergent evolution of these subsites, the distinguishing structural features determining the overall carbohydrate specificities are located in extended binding site regions. In pLT, Arg13 is thus found to play a crucial role in enhancing the affinity not only for N-acetyllactosamine-terminated structures but also for GM1 as compared to human LT (hLT) and CT. The physiological relevance of the binding of N-acetyllactosamine-containing glycoconjugates to LT and ECORL is briefly discussed.

CT      Amino Sugars: ME, metabolism  
         Animals  
         \*Antigens, CD  
         \*Bacterial Toxins: ME, metabolism  
         Crystallography, X-Ray  
         \*Enterotoxins: ME, metabolism  
         \*Erythrina: ME, metabolism  
         \*Escherichia coli: ME, metabolism  
         \*Escherichia coli Proteins  
         \*Galactose: ME, metabolism  
         Humans  
         Hydrogen Bonding  
         Isotope Labeling  
         \*Lactosylceramides: ME, metabolism  
         \*Lectins: ME, metabolism  
         Ligands  
         Magnetic Resonance Spectroscopy  
         Models, Molecular  
         Plant Lectins  
         \*Plants, Medicinal  
         Protein Conformation  
         Structure-Activity Relationship  
         Swine

RN      26566-61-0 (Galactose); 32181-59-2 (N-acetyllactosamine); 4682-48-8 (CDw17 antigen)

CN      0 (Amino Sugars); 0 (Antigens, CD); 0 (Bacterial Toxins); 0 (Enterotoxins); 0 (Escherichia coli Proteins); 0 (Lactosylceramides); 0 (Lectins); 0 (Ligands); 0 (Plant Lectins); 0 (enterotoxin LT)

L44     ANSWER 3 OF 7      MEDLINE on STN  
 AN     97403073      MEDLINE  
 DN     PubMed ID: 9258442  
 TI     Immobilization of reducing sugars as toxin binding agents.  
 AU     Nilsson U J; Heerze L D; Liu Y C; Armstrong G D; Palcic M M; Hindsgaul O  
 CS     Department of Chemistry, University of Alberta, Edmonton, Canada.  
 SO     Bioconjugate chemistry, (1997 Jul-Aug) Vol. 8, No. 4, pp. 466-71.  
        Journal code: 9010319. ISSN: 1043-1802.  
 CY     United States  
 DT     Journal; Article; (JOURNAL ARTICLE)  
        (RESEARCH SUPPORT, NON-U.S. GOV'T)  
 LA     English

FS Priority Journals  
 EM 199710  
 ED Entered STN: 24 Oct 1997  
 Last Updated on STN: 24 Oct 1997  
 Entered Medline: 10 Oct 1997  
 AB A simple and economical procedure for the attachment of reducing sugars to aminated solid supports has been developed. Reaction of the amino groups on the solid support with p-nitrophenyl chloroformate, followed by 1,6-hexanediamine, yields a chain-extended amine to which reducing sugars can be attached while remaining accessible to macromolecules. Immobilization of the reducing sugars involves a simple incubation followed by trapping of the resulting glycosylamine with acetic anhydride and recovery of the unreacted sugar by filtration. This technique was used to immobilize lactose and **sialyllactose** onto silylaminated Chromosorb P, producing solid supports that effectively neutralized the activity of cholera toxin from *Vibrio cholerae* and heat-labile **enterotoxin** of **enterotoxigenic Escherichia coli**. The general applicability of such solid supports for toxin neutralization was further demonstrated by immobilization of the enzymatically synthesized alpha Gal(1-3) beta Gal(1-4)Glc trisaccharide, which produced a support that efficiently neutralized toxin A of *Clostridium difficile*. The results from this study suggest that these solid supports have the potential to serve as inexpensive therapeutics for bacterial toxin-mediated **diarrheal** diseases.  
 CT Animals  
 \*Bacterial Toxins: ME, metabolism  
 CHO Cells  
 Carbohydrate Sequence  
 \*Cholera Toxin: ME, metabolism  
 Cricetinae  
 \*Enterotoxins: ME, metabolism  
 Escherichia coli: CH, chemistry  
 \*Escherichia coli Proteins  
 Molecular Sequence Data  
 \*Oligosaccharides: CH, chemistry  
 Oligosaccharides: ME, metabolism  
 Oxidation-Reduction  
 Protein Binding  
 RN 9012-63-9 (Cholera Toxin)  
 CN 0 (Bacterial Toxins); 0 (**Enterotoxins**); 0 (**Escherichia coli Proteins**); 0 (Oligosaccharides); 0 (**enterotoxin LT**); 0 (tcdA protein, *Clostridium difficile*)  
 L44 ANSWER 4 OF 7 MEDLINE on STN  
 AN 95252586 MEDLINE  
 DN PubMed ID: 7766178  
 TI Inhibition of cholera toxin by human milk fractions and **sialyllactose**.  
 AU Idota T; Kawakami H; Murakami Y; Sugawara M  
 CS Technical Research Institute, Snow Brand Milk Products Co., Ltd., Saitama, Japan.  
 SO Bioscience, biotechnology, and biochemistry, (1995 Mar) Vol. 59, No. 3, pp. 417-9.  
 Journal code: 9205717. ISSN: 0916-8451.  
 CY Japan  
 DT (IN VITRO)  
 Journal; Article; (JOURNAL ARTICLE)  
 LA English  
 FS Biotechnology  
 EM 199506

ED Entered STN: 9 Aug 1995  
 Last Updated on STN: 9 Aug 1995  
 Entered Medline: 8 Jun 1995

AB The effects of human milk fractions on cholera toxin B subunit binding to monosialoganglioside 1 (GM1) were investigated. Human milk, human defatted milk, whey, and a low-molecular-weight fraction of human milk inhibited the binding, but casein did not inhibit it. The inhibitory activity of whey from bovine-milk-based infant formula was less than that of whey from human milk. Differences in composition between human and bovine whey seemed to influence the extent of the inhibitory activity. Sialylated oligosaccharides were considered to be the possible components that inhibited cholera toxin. The effects of **sialyllactose**, a predominant sialylated component of human milk, on cholera toxin-induced diarrhea were investigated by the rabbit intestinal loop method. **Sialyllactose** inhibited the cholera toxin inducing fluid accumulation, although neither sialic acid nor lactose had an effect on it. The results suggest that **sialyllactose** is responsible for the inhibitory activity of milk on cholera toxin.

CT Check Tags: Male  
 Animals  
 Binding, Competitive: DE, drug effects  
 Body Fluids: DE, drug effects  
 \***Cholera Toxin: AI, antagonists & inhibitors**  
 G(M1) Ganglioside: PD, pharmacology  
 Humans  
 Intestines: DE, drug effects  
 Intestines: ME, metabolism  
 \***Lactose: AA, analogs & derivatives**  
 Lactose: PD, pharmacology  
 \***Milk, Human: CH, chemistry**  
 Oligosaccharides: IP, isolation & purification  
 Oligosaccharides: PD, pharmacology  
 Rabbits  
 \***Sialic Acids: PD, pharmacology**

RN 35890-38-1 (**N-acetylneuraminyllactose**); 37758-47-7 (G(M1)  
 Ganglioside); 63-42-3 (Lactose); 9012-63-9 (Cholera Toxin)

CN 0 (Oligosaccharides); 0 (Sialic Acids)

L44 ANSWER 5 OF 7 MEDLINE on STN  
 AN 93293800 MEDLINE  
 DN PubMed ID: 8514738  
 TI Postnatal change of pig intestinal ganglioside bound by **Escherichia coli** with K99 fimbriae.  
 AU Yuyama Y; Yoshimatsu K; Ono E; Saito M; Naiki M  
 CS Faculty of Veterinary Medicine, Hokkaido University.  
 SO Journal of biochemistry, (1993 Apr) Vol. 113, No. 4, pp. 488-92.  
 Journal code: 0376600. ISSN: 0021-924X.  
 CY Japan  
 DT Journal; Article; (JOURNAL ARTICLE)  
 (RESEARCH SUPPORT, NON-U.S. GOV'T)  
 LA English  
 FS Priority Journals  
 EM 199307  
 ED Entered STN: 6 Aug 1993  
 Last Updated on STN: 6 Aug 1993  
 Entered Medline: 22 Jul 1993

AB **Enterotoxigenic Escherichia coli** possessing K99 fimbriae (**E. coli** K99) causes **diarrhea** in piglets of less than 1 week old. The first stage of the bacterial infection is adhesion by the fimbriae on the small intestinal mucosa and

the adhesion is followed by colony formation. K99 fimbriae bind specifically to **N-glycolylneuraminyllactosylceramide**, GM3(NeuGc) [Ono, E. et al. (1989) *Infect. Immun.* 57, 907-911]. We examined the postnatal change of the content and the molecular species of GM3(NeuGc) in the small intestinal mucosa of 0- to 14-day-old piglets and adult pigs. GM3(NeuGc) was a major ganglioside of piglet intestinal mucosa. GM3(NeuGc) content was maximal at birth and gradually decreased to 1/16 in adult animals (5 months old). The **ceramide** moiety of piglet intestinal GM3(NeuGc) was characterized by the presence of 2-hydroxylated palmitic acid. <sup>125</sup>I-labeled bacteria strongly bound to GM3(NeuGc) containing 2-hydroxylated palmitic acid and phytosphingosine compared with GM3(NeuGc) containing any other **ceramide** moiety. The time when this particular GM3(NeuGc) appears coincides with the time that the infection occurs, and it may explain the susceptibility of newborn piglets to *E. coli* K99 infection.

CT Animals  
 Animals, Newborn  
 Chromatography, Thin Layer  
 Enterotoxins  
 \*Escherichia coli: PY, pathogenicity  
 \*Escherichia coli Infections: MI, microbiology  
 \*G(M3) Ganglioside: AA, analogs & derivatives  
 G(M3) Ganglioside: ME, metabolism  
 Gangliosides: ME, metabolism  
 \*Intestinal Diseases: MI, microbiology  
 \*Intestinal Mucosa: ME, metabolism  
 \*Intestinal Mucosa: MI, microbiology  
 Intestine, Small: ME, metabolism  
 Intestine, Small: MI, microbiology  
 Swine

RN 69345-49-9 (**N-glycolylneuraminyllactosylceramide**)  
 CN 0 (Enterotoxins); 0 (G(M3) Ganglioside); 0 (Gangliosides)

L44 ANSWER 6 OF 7 MEDLINE on STN  
 AN 89339746 MEDLINE  
 DN PubMed ID: 2503449  
 TI Hemagglutinating properties of *Shigella dysenteriae* type 1 and other *Shigella* species.  
 AU Qadri F; Haq S; Ciznar I  
 CS Laboratory Sciences Division, International Centre for Diarrhoeal Disease and Research, Bangladesh.  
 SO Infection and immunity, (1989 Sep) Vol. 57, No. 9, pp. 2909-11.  
 Journal code: 0246127. ISSN: 0019-9567.  
 CY United States  
 DT Journal; Article; (JOURNAL ARTICLE)  
 LA English  
 FS Priority Journals  
 EM 198909  
 ED Entered STN: 9 Mar 1990  
 Last Updated on STN: 9 Mar 1990  
 Entered Medline: 15 Sep 1989  
 AB Strains of *Shigella dysenteriae* type 1 cultured in Casamino Acids-yeast extract broth medium in the presence of 1 mM calcium chloride at 37 degrees C for 22 h induced hemagglutination of erythrocytes that was inhibited by **N-acetylneuraminic acid**, **N-acetylneuraminic-lactose**, and alpha 1-glycoprotein. The hemagglutination was heat labile, and the absence of cell-surface appendages suggested a nonfimbrial adhesin(s). Under the same conditions, strains of *Shigella flexneri* (types 1a, 1b, 2a, and 2b) showed **N-acetylneuraminic**

acid-resistant hemagglutination of erythrocytes.

CT Animals

Cattle

Culture Media: AN, analysis

Guinea Pigs

Haplorhini

\*Hemagglutination Tests

Humans

Rabbits

Sheep

*Shigella dysenteriae: GD, growth & development*

*\*Shigella dysenteriae: IM, immunology*

Swine

CN 0 (Culture Media)

L44 ANSWER 7 OF 7 MEDLINE on STN

AN 79216779 MEDLINE

DN PubMed ID: 222809

TI Gangliosides sensitize unresponsive fibroblasts to *Escherichia coli* heat-labile **enterotoxin**.

AU Moss J; Garrison S; Fishman P H; Richardson S H

SO The Journal of clinical investigation, (1979 Aug) Vol. 64, No. 2, pp. 381-4.

Journal code: 7802877. ISSN: 0021-9738.

CY United States

DT Journal; Article; (JOURNAL ARTICLE)  
(RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)

LA English

FS Abridged Index Medicus Journals; Priority Journals

EM 197909

ED Entered STN: 15 Mar 1990  
Last Updated on STN: 15 Mar 1990  
Entered Medline: 25 Sep 1979

AB Chemically transformed mouse fibroblasts did not raise their cyclic AMP level in response to *Escherichia coli* heat-labile **enterotoxin**. These fibroblasts did, however, incorporate exogenous mono-, di-, and trisialogangliosides. After the uptake of monosialoganglioside galactosyl-N-acetylgalactosaminyl-[N-acetylneuraminyll]-galactosylglucosylceramide (GM1), the cells responded to *E. coli* heat-labile **enterotoxin**. The di- and trisialogangliosides were considerably less effective. GM1, the putative cholera toxin (choleragen) receptor, has been implicated previously as the receptor for *E. coli* heat-labile **enterotoxin** based on the ability of the free ganglioside to inhibit the effects of toxin. This investigation establishes that the ganglioside, when incorporated into fibroblasts, serves a functional role in mediating the responsiveness to the toxin.

CT Animals

Cell Line

*Cholera Toxin: PD, pharmacology*

\*Cyclic AMP: ME, metabolism

*\*Enterotoxins: PD, pharmacology*

*\*Escherichia coli*

*Fibroblasts: DE, drug effects*

\*Fibroblasts: ME, metabolism

\*Gangliosides: PD, pharmacology

*Heat*

*Mice*

RN 60-92-4 (Cyclic AMP); 9012-63-9 (Cholera Toxin)

CN 0 (**Enterotoxins**); 0 (**Gangliosides**)

=> d 158 bib ab tot

L58 ANSWER 1 OF 16 MEDLINE on STN  
 AN 2007131051 MEDLINE  
 DN PubMed ID: 17268859  
 TI Conjugation of oligosaccharides by reductive amination to amine modified chondroitin oligomer and gamma-cyclodextrin.  
 AU Weikkolainen Krista; Aitio Olli; Blomqvist Maria; Natunen Jari; Helin Jari  
 CS Department of Biological and Environmental Sciences, University of Helsinki, P. O. Box 56, 00014, Helsinki, Finland.  
 SO Glycoconjugate journal, (2007 Apr) Vol. 24, No. 2-3, pp. 157-65.  
 Electronic Publication: 2007-02-01.  
 Journal code: 8603310. ISSN: 0282-0080.  
 CY United States  
 DT Journal; Article; (JOURNAL ARTICLE)  
 (RESEARCH SUPPORT, NON-U.S. GOV'T)  
 LA English  
 FS Priority Journals  
 EM 200711  
 ED Entered STN: 3 Mar 2007  
 Last Updated on STN: 9 Nov 2007  
 Entered Medline: 8 Nov 2007  
 AB Carbohydrates present on cell surfaces participate in numerous biological recognition phenomena including cell-cell interactions, cancer metastasis and pathogen invasion. Therefore, synthetic carbohydrates have a potential to act as pharmaceutical substances for treatment of various pathological phenomena by inhibiting specifically the interaction between cell surface carbohydrates and their protein receptors (lectins). However, the inherently low affinity of carbohydrate-protein interactions has often been an obstacle for successful generation of carbohydrate based pharmaceuticals. Multivalent glycoconjugates, i.e. structures carrying several copies of the active carbohydrate sequence in a carrier molecule, have been constructed to overcome this problem. Here we present two novel types of multivalent carbohydrate conjugates based on chondroitin oligomer and cyclodextrin carriers. These carriers were modified to express primary amino groups, and oligosaccharides were then bound to carrier molecules by reductive amination. Multivalent conjugates were produced using the human milk type oligosaccharides LNDFH I (Lewis-*b* hexasaccharide), LNnT, and GlcNAcbeta1-3Galbeta1-4GlcNAcbeta1-3Galbeta1-4Glc.

L58 ANSWER 2 OF 16 MEDLINE on STN  
 AN 2006599489 MEDLINE  
 DN PubMed ID: 16880000  
 TI Binding of *Haemophilus ducreyi* to carbohydrate receptors is mediated by the 58.5-kDa GroEL heat shock protein.  
 AU Pantzar Martina; Teneberg Susann; Lagergard Teresa  
 CS Institute of Biomedicine, Department of Microbiology and Immunology, Goteborg University, PO Box 435, SE-40530, Goteborg, Sweden.  
 SO Microbes and infection / Institut Pasteur, (2006 Aug) Vol. 8, No. 9-10, pp. 2452-8. Electronic Publication: 2006-07-07.  
 Journal code: 100883508. ISSN: 1286-4579.  
 CY France  
 DT Journal; Article; (JOURNAL ARTICLE)  
 (RESEARCH SUPPORT, NON-U.S. GOV'T)  
 LA English  
 FS Priority Journals

EM 200702  
 ED Entered STN: 11 Oct 2006  
 Last Updated on STN: 14 Feb 2007  
 Entered Medline: 13 Feb 2007  
 AB The bacterium *Haemophilus ducreyi* causes the sexually transmitted disease chancroid, which is characterized by the appearance of mucocutaneous, persistent ulcers on the external genitals. To identify carbohydrate receptors that mediate the attachment of this pathogen to host cells, we investigated the binding of 35S-methionine-labeled *H. ducreyi* strains to a panel of defined glycosphingolipids that were separated on thin layer chromatography plates. *H. ducreyi* bound to lactosylceramide, gangliotriaosylceramide, gangliotetraosylceramide, the GM3 ganglioside, and sulfatide. To elucidate the role of the surface-located 58.5-kDa GroEL heat shock protein (HSP) of *H. ducreyi* in attachment, we investigated the binding of purified HSP to the same panel of glycosphingolipids. Our results suggest that the 58.5-kDa GroEL HSP of *H. ducreyi* is responsible for the attachment of this bacterium to the majority of the tested glycosphingolipids, and thus represents a potential bacterial adhesin.

L58 ANSWER 3 OF 16 MEDLINE on STN  
 AN 2006283542 MEDLINE  
 DN PubMed ID: 16714580  
 TI The major subunit, CfaB, of colonization factor antigen i from **enterotoxigenic Escherichia coli** is a glycosphingolipid binding protein.  
 AU Jansson Lena; Tobias Joshua; Lebens Michael; Svennerholm Ann-Mari; **Teneberg Susann**  
 CS Department of Medical Biochemistry, Institute of Biomedicine, Goteborg University, P.O. Box 440, S-405 30 Goteborg, Sweden.  
 SO Infection and immunity, (2006 Jun) Vol. 74, No. 6, pp. 3488-97.  
 Journal code: 0246127. ISSN: 0019-9567.  
 CY United States  
 DT Journal; Article; (JOURNAL ARTICLE)  
 (RESEARCH SUPPORT, NON-U.S. GOV'T)  
 LA English  
 FS Priority Journals  
 EM 200606  
 ED Entered STN: 23 May 2006  
 Last Updated on STN: 16 Jun 2006  
 Entered Medline: 15 Jun 2006  
 AB Bacterial adherence to mucosal surfaces is an important virulence trait of pathogenic bacteria. Adhesion of **enterotoxigenic Escherichia coli** (ETEC) to the intestine is mediated by a number of antigenically distinct colonization factors (CFs). One of the most common CFs is CFA/I. This has a fimbrial structure composed of a major repeating subunit, CfaB, and a single tip subunit, CfaE. The potential carbohydrate recognition by CFA/I was investigated by binding CFA/I-fimbriated bacteria and purified CFA/I fimbriae to a large number of variant glycosphingolipids separated on thin-layer chromatograms. For both fimbriated bacteria and purified fimbriae, specific interactions could be identified with a number of nonacid glycosphingolipids. These included glucosylceramide, lactosylceramide with phytosphingosine and/or hydroxy fatty acids, **neolactotetraosylceramide**, gangliotriaosylceramide, gangliotetraosylceramide, the H5 type 2 pentaglycosylceramide, the Lea-5 glycosphingolipid, the Lex-5 glycosphingolipid, and the Ley-6 glycosphingolipid. These glycosphingolipids were also recognized by recombinant **E. coli** expressing CFA/I in the absence of tip protein CfaE, as well as by purified fimbriae from the same strain. This demonstrates that the

glycosphingolipid-binding capacity of CFA/I resides in the major CfaB subunit.

L58 ANSWER 4 OF 16 MEDLINE on STN  
 AN 2004041592 MEDLINE  
 DN PubMed ID: 14576169  
 TI Carbohydrate recognition by enterohemorrhagic **Escherichia coli**: characterization of a novel glycosphingolipid from cat small intestine.  
 AU **Teneberg Susann; Angstrom Jonas; Ljungh Asa**  
 CS Institute of Medical Biochemistry, Goteborg University, SE 405 30 Goteborg, Sweden.. susann.teneberg@medkem.gu.se  
 SO Glycobiology, (2004 Feb) Vol. 14, No. 2, pp. 187-96. Electronic Publication: 2003-10-23.  
 Journal code: 9104124. ISSN: 0959-6658.  
 CY England: United Kingdom  
 DT Journal; Article; (JOURNAL ARTICLE)  
 (RESEARCH SUPPORT, NON-U.S. GOV'T)  
 LA English  
 FS Priority Journals  
 EM 200410  
 ED Entered STN: 27 Jan 2004  
 Last Updated on STN: 20 Oct 2004  
 Entered Medline: 19 Oct 2004  
 AB A key virulence trait of pathogenic bacteria is the ability to bind to receptors on mucosal cells. Here the potential glycosphingolipid receptors of enterohemorrhagic **Escherichia coli** were examined by binding of 35S-labeled bacteria to glycosphingolipids on thin-layer chromatograms. Thereby a selective interaction with two nonacid glycosphingolipids of cat small intestinal epithelium was found. The binding-active glycosphingolipids were isolated and, on the basis of mass spectrometry, proton NMR spectroscopy, and degradation studies, identified as Galalpha3Galbeta4Glcbeta1Cer (isoglobotriaosylceramide) and Galalpha3Galalpha3Galbeta4Glcbeta1Cer. The latter glycosphingolipid has not been described before. The interaction was not based on terminal Galalpha3 because the bacteria did not recognize the structurally related glycosphingolipids Galalpha3Galalpha4Galbeta4Glcbeta1Cer and Galalpha3Galbeta4GlcNAcbeta3Galbeta4Glcbeta1Cer (B5 glycosphingolipid). However, further binding assays using reference glycosphingolipids showed that the enterohemorrhagic **E. coli** also bound to lactosylceramide with phytosphingosine and/or hydroxy fatty acids, suggesting that the minimal structural element recognized is a correctly presented lactosyl unit. Further binding of **neolactotetraosylceramide**, lactotetraosylceramide, the Le(a)-5 glycosphingolipid, as well as a weak binding to gangliotriaosylceramide and gangliotetraosylceramide, was found in analogy with binding patterns that previously have been described for other bacteria classified as lactosylceramide-binding.

L58 ANSWER 5 OF 16 MEDLINE on STN  
 AN 2001698041 MEDLINE  
 DN PubMed ID: 11744628  
 TI **Helicobacter pylori**-binding gangliosides of human gastric adenocarcinoma.  
 AU **Roche N; Larsson T; Angstrom J; Teneberg S**  
 CS Institute of Medical Biochemistry, Goteborg University, P.O. Box 440, SE 405 30 Goteborg, Sweden.  
 SO Glycobiology, (2001 Nov) Vol. 11, No. 11, pp. 935-44.  
 Journal code: 9104124. ISSN: 0959-6658.  
 CY England: United Kingdom  
 DT (IN VITRO)

Journal; Article; (JOURNAL ARTICLE)  
(RESEARCH SUPPORT, NON-U.S. GOV'T)

LA English  
FS Priority Journals  
EM 200203  
ED Entered STN: 18 Dec 2001  
Last Updated on STN: 26 Mar 2002  
Entered Medline: 25 Mar 2002  
AB Acidic and neutral glycosphingolipids were isolated from a human gastric adenocarcinoma, and binding of *Helicobacter pylori* to the isolated glycosphingolipids was assessed using the chromatogram binding assay. The isolated glycosphingolipids were characterized using fast atom bombardment mass spectrometry and by binding of antibodies and lectins. The predominating neutral glycosphingolipids were found to migrate in the di- to tetraglycosylceramide regions as revealed by anisaldehyde staining and detection with lectins. No binding of *H. pylori* to these compounds was obtained. The most abundant acidic glycosphingolipids, migrating as the GM3 ganglioside and sialyl-neolactotetraosylceramide, were not recognized by the bacteria. Instead, *H. pylori* selectively interacted with slow-migrating, low abundant gangliosides not detected by anisaldehyde staining. Binding-active gangliosides were isolated and characterized by mass spectrometry, proton nuclear magnetic resonance, and lectin binding as sialyl-neolactohexaosylceramide (NeuAcalpha3Galbeta4GlcNAcbeta3Galbeta4GlcNAcbeta3Galbeta4GlcNAcbeta3Galbeta4GlcNAcbeta3Galbeta4GlcNAcbeta3Galbeta4GlcNAcbeta1Cer) and sialyl-neolactooctaosylceramide (NeuAcalpha3Galbeta4GlcNAcbeta3Galbeta4GlcNAcbeta3Galbeta4GlcNAcbeta3Galbeta4GlcNAcbeta1Cer).

L58 ANSWER 6 OF 16 MEDLINE on STN  
AN 2001111860 MEDLINE  
DN PubMed ID: 11056399  
TI Isolectins from *Solanum tuberosum* with different detailed carbohydrate binding specificities: unexpected recognition of lactosylceramide by N-acetyllactosamine-binding lectins.  
AU Ciopraga J; Angstrom J; Bergstrom J; Larsson T; Karlsson N; Motas C; Gozia O; Teneberg S  
CS Institute of Biochemistry, Romanian Academy, P.O. Box 78 200 Bucharest, 2, Romania.  
SO Journal of biochemistry, (2000 Nov) Vol. 128, No. 5, pp. 855-67.  
Journal code: 0376600. ISSN: 0021-924X.  
CY Japan  
DT Journal; Article; (JOURNAL ARTICLE)  
(RESEARCH SUPPORT, NON-U.S. GOV'T)  
LA English  
FS Priority Journals  
EM 200102  
ED Entered STN: 22 Mar 2001  
Last Updated on STN: 18 Dec 2002  
Entered Medline: 8 Feb 2001  
AB Glycosphingolipid recognition by two isolectins from *Solanum tuberosum* was compared by the chromatogram binding assay. One lectin (PL-I) was isolated from potato tubers by affinity chromatography, and identified by MALDI-TOF mass spectrometry as a homodimer with a subunit molecular mass of 63,000. The other (PL-II) was a commercial lectin, characterized as two homodimeric isolectins with subunit molecular masses of 52,000 and 55,000, respectively. Both lectins recognized N-acetyllactosamine-containing glycosphingolipids, but the fine details of their carbohydrate binding specificities differed. PL-II preferentially bound to glycosphingolipids with N-acetyllactosamine branches, as Galbeta4GlcNAcbeta6(Galbeta4GlcNAcbeta3)Galbeta4Glcbeta1Cer. PL-I also recognized this glycosphingolipid, but bound equally well to the linear

glycosphingolipid Galbeta4GlcNAcbeta3Galbeta4GlcNAcbeta3Galbeta4Glcbeta1Cer. **Neolactotetraosylceramide** and the B5 pentaglycosylceramide were also bound by PL-I, while other glycosphingolipids with only one N-acetyllactosamine unit were non-binding. Surprisingly, both lectins also bound to lactosylceramide, with an absolute requirement for sphingosine and non-hydroxy fatty acids. The inhibition of binding to both lactosylceramide and N-acetyllactosamine-containing glycosphingolipids by N-acetylchitotetraose suggests that lactosylceramide is also accommodated within the N-acetylchitotetraose/N-acetyllactosamine-binding sites of the lectins. Through docking of glycosphingolipids onto a three-dimensional model of the PL-I hevein binding domain, a Galbeta4GlcNAcbeta3Galbeta4 binding epitope was defined. Furthermore, direct involvement of the ceramide in the binding of lactosylceramide was suggested.

L58 ANSWER 7 OF 16 MEDLINE on STN  
 AN 2001110159 MEDLINE  
 DN PubMed ID: 11087709  
 TI Inhibition of nonopsonic *Helicobacter pylori*-induced activation of human neutrophils by sialylated oligosaccharides.  
 AU **Teneberg S**; Jurstrand M; **Karlsson K A**; Danielsson D  
 CS Institute of Medical Biochemistry, Goteborg University, P.O. Box 440, SE 405 30 Goteborg, Sweden.  
 SO Glycobiology, (2000 Nov) Vol. 10, No. 11, pp. 1171-81.  
 Journal code: 9104124. ISSN: 0959-6658.  
 CY ENGLAND: United Kingdom  
 DT (IN VITRO)  
 Journal; Article; (JOURNAL ARTICLE)  
 (RESEARCH SUPPORT, NON-U.S. GOV'T)  
 LA English  
 FS Priority Journals  
 EM 200102  
 ED Entered STN: 22 Mar 2001  
 Last Updated on STN: 22 Mar 2001  
 Entered Medline: 2 Feb 2001  
 AB Certain strains of *Helicobacter pylori* have nonopsonic neutrophil-activating capacity. Some *H. pylori* strains and the neutrophil-activating protein of *H. pylori* (HPNAP) bind selectively to gangliosides of human neutrophils. To determine if there is a relationship between the neutrophil-activating capacity and the ganglioside-binding ability, a number of *H. pylori* strains, and HPNAP, were incubated with oligosaccharides, and the effects on the oxidative burst of subsequently challenged neutrophils was measured by chemiluminescence and flow cytometry. Both by chemiluminescence and flow cytometry a reduced response was obtained by incubation of *H. pylori* with sialic acid-terminated oligosaccharides, whereas lactose had no effect. The reductions obtained with different sialylated oligosaccharides varied to some extent between the *H. pylori* strains, but in general 3'-**sialyllactosamine** was the most efficient inhibitor. Challenge of neutrophils with HPNAP gave no response in the chemiluminescence assay, and a delayed moderate response with flow cytometry. Preincubation of the protein with 3'-**sialyllactosamine** gave a slight reduction of the response, while 3'-**sialyllactose** had no effect. The current results suggest that the nonopsonic *H. pylori*-induced activation of neutrophils occurs by lectinophagocytosis, the recognition of sialylated glycoconjugates on the neutrophil cell surface by a bacterial adhesin leads to phagocytosis and an oxidative burst with the production of reactive oxygen metabolites.

L58 ANSWER 8 OF 16 MEDLINE on STN

AN 2001060702 MEDLINE  
 DN PubMed ID: 10965049  
 TI Common architecture of the primary galactose binding sites of *Erythrina corallodendron* lectin and heat-labile **enterotoxin** from *Escherichia coli* in relation to the binding of branched **neolactohexaosylceramide**.  
 AU Teneberg S; Berntsson A; Angstrom J  
 CS Institute of Medical Biochemistry, Goteborg University, P.O. Box SE 405 30 Goteborg, Sweden.  
 SO Journal of biochemistry, (2000 Sep) Vol. 128, No. 3, pp. 481-91.  
 CY Japan  
 DT (COMPARATIVE STUDY)  
 Journal; Article; (JOURNAL ARTICLE)  
 (RESEARCH SUPPORT, NON-U.S. GOV'T)  
 LA English  
 FS Priority Journals  
 EM 200012  
 ED Entered STN: 22 Mar 2001  
 Last Updated on STN: 18 Dec 2002  
 Entered Medline: 22 Dec 2000  
 AB The heat-labile **enterotoxin** from *Escherichia coli* (LT) is responsible for so-called traveller's **diarrhea** and is closely related to the cholera toxin (CT). Toxin binding to GM1 at the epithelial cell surface of the small intestine initiates the subsequent **diarrheal** disease. However, LT has a broader receptor specificity than CT in that it also binds to N-acetyllactosamine-terminated structures. The unrelated lectin from *Erythrina corallodendron* (ECorL) shares this latter binding property. The findings that both ECorL and porcine LT (pLT) bind to lactose as well as to **neolactotetraosylceramide** suggests a common structural theme in their respective primary binding sites. Superimposing the terminal galactose of the lactoses in the respective crystal structures of pLT and ECorL reveals striking structural similarities around the galactose despite the lack of sequence and folding homology, whereas the interactions of the penultimate GlcNAcb3 in the **neolactotetraosylceramide** differ. The binding of branched **neolactohexaosylceramide** to either protein reveals an enhanced affinity relative to **neolactotetraosylceramide**. The b3-linked branch is found to bind to the primary Gal binding pocket of both proteins, whereas the b6-linked branch outside this site provides additional interactions in accordance with the higher binding affinities found for this compound. While the remarkable architectural similarities of the primary galactose binding sites of pLT and ECorL point to a convergent evolution of these subsites, the distinguishing structural features determining the overall carbohydrate specificities are located in extended binding site regions. In pLT, Arg13 is thus found to play a crucial role in enhancing the affinity not only for N-acetyllactosamine-terminated structures but also for GM1 as compared to human LT (hLT) and CT. The physiological relevance of the binding of N-acetyllactosamine-containing glycoconjugates to LT and ECorL is briefly discussed.

L58 ANSWER 9 OF 16 MEDLINE on STN  
 AN 1999054731 MEDLINE  
 DN PubMed ID: 9832619  
 TI Glycosphingolipid binding specificities of *Neisseria meningitidis* and *Haemophilus influenzae*: detection, isolation, and characterization of a binding-active glycosphingolipid from human oropharyngeal epithelium.  
 AU Hugosson S; Angstrom J; Olsson B M; Bergstrom J; Fredlund H; Olcen P; Teneberg S

CS Department of Otorhinolaryngology, Orebro Medical Center Hospital, Orebro, Sweden.  
 SO Journal of biochemistry, (1998 Dec 1) Vol. 124, No. 6, pp. 1138-52.  
 Journal code: 0376600. ISSN: 0021-924X.  
 CY Japan  
 DT (COMPARATIVE STUDY)  
 Journal; Article; (JOURNAL ARTICLE)  
 (RESEARCH SUPPORT, NON-U.S. GOV'T)  
 LA English  
 FS Priority Journals  
 EM 199903  
 ED Entered STN: 24 Mar 1999  
 Last Updated on STN: 24 Mar 1999  
 Entered Medline: 9 Mar 1999  
 AB The glycosphingolipid binding specificities of *Haemophilus influenzae* and *Neisseria meningitidis* were investigated as to the binding of radiolabeled bacteria to glycosphingolipids on thin-layer chromatograms. Thereby, similar binding profiles, for the binding of the two bacteria to lactosylceramide, isoglobotriaosylceramide, gangliotriaosylceramide, gangliotetraosylceramide, lactotetraosylceramide, **neolactotetraosylceramide**, and sialylneolactohexaosylceramide, were obtained. On a closer view the binding preferences of the bacteria could be differentiated into three groups. The first specificity is recognition of lactosylceramide. The second specificity is binding to gangliotriaosylceramide and gangliotetraosylceramide, since conversion of the acetamido group of the N-acetylgalactosamine of gangliotriaosylceramide and gangliotetraosylceramide to an amine prevented the binding of the bacteria, and thus the binding to these two glycosphingolipids represents a separate specificity from lactosylceramide recognition. Preincubation of *H. influenzae* with **neolactotetraose** inhibited the binding to **neolactotetraosylceramide**, while the binding to lactosylceramide, gangliotetraosylceramide, or lactotetraosylceramide was unaffected. Thus, the third binding specificity is represented by **neolactotetraosylceramide**, and involves recognition of other neolacto series glycosphingolipids with linear N-acetyllactosamine chains, such as sialyl-neolactohexaosylceramide. The relevance of the detected binding specificities for adhesion to target cells was addressed as to the binding of the bacteria to glycosphingolipids from human granulocytes, epithelial cells of human nasopharyngeal tonsils and human plexus choroideus. Binding-active **neolactotetraosylceramide** was thereby detected in human granulocytes and the oropharyngeal epithelium.

L58 ANSWER 10 OF 16 MEDLINE on STN  
 AN 97323395 MEDLINE  
 DN PubMed ID: 9179843  
 TI Structural basis for differential receptor binding of cholera and *Escherichia coli* heat-labile toxins: influence of heterologous amino acid substitutions in the cholera B-subunit.  
 AU Backstrom M; Shahabi V; Johansson S; **Teneberg S**; Kjellberg A; **Miller-Podraza H**; Holmgren J; Lebens M  
 CS Department of Medical Microbiology and Immunology, Goteborg University, Sweden.  
 SO Molecular microbiology, (1997 May) Vol. 24, No. 3, pp. 489-97.  
 Journal code: 8712028. ISSN: 0950-382X.  
 CY ENGLAND: United Kingdom  
 DT (COMPARATIVE STUDY)  
 (IN VITRO)  
 Journal; Article; (JOURNAL ARTICLE)  
 (RESEARCH SUPPORT, NON-U.S. GOV'T)

LA English  
 FS Priority Journals  
 EM 199708  
 ED Entered STN: 25 Aug 1997  
 Last Updated on STN: 25 Aug 1997  
 Entered Medline: 13 Aug 1997  
 AB The closely related B-subunits of cholera toxin (CTB) and **Escherichia coli** heat-labile **enterotoxin** (LTB) both bind strongly to GM1 ganglioside receptors but LTB can also bind to additional glycolipids and glycoproteins. A number of mutant CT B-subunits were generated by substituting CTB amino acids with those at the corresponding positions in LTB. These were used to investigate the influence of specific residues on receptor-binding specificity. A mutated CTB protein containing the first 25 residues of LTB in combination with LTB residues at positions 94 and 95, bound to the same extent as native LTB to both delipidized rabbit intestinal cell membranes, complex glycosphingolipids (polyglycosylceramides) and **neolactotetraosylceramide**, but not to non-GM1 intestinal glycosphingolipids. In contrast, when LTB amino acid substitutions in the 1-25 region were combined with those in the 75-83 region, a binding as strong as that of LTB to intestinal glycosphingolipids was observed. In addition, a mutant LTB with a single Gly-33-->Asp substitution that completely lacked affinity for both GM1 and non-GM1 glycosphingolipids could still bind to receptors in the intestinal cell membranes and to polyglycosylceramides. We conclude that the extra, non-GM1 receptors for LTB consist of both sialylated and non-sialylated glycoconjugates, and that the binding to either class of receptors is influenced by different amino acid residues within the protein.

L58 ANSWER 11 OF 16 MEDLINE on STN  
 AN 96375687 MEDLINE  
 DN PubMed ID: 8781976  
 TI Recognition of glycoconjugates by *Helicobacter pylori*: an apparently high-affinity binding of human polyglycosylceramides, a second sialic acid-based specificity.  
 AU **Miller-Podraza H; Milh M A; Bergstrom J; Karlsson K A**  
 CS Department of Medical Biochemistry, Goteborg University, Sweden.  
 SO Glycoconjugate journal, (1996 Jun) Vol. 13, No. 3, pp. 453-60.  
 Journal code: 8603310. ISSN: 0282-0080.  
 CY ENGLAND: United Kingdom  
 DT Journal; Article; (JOURNAL ARTICLE)  
 (RESEARCH SUPPORT, NON-U.S. GOV'T)  
 LA English  
 FS Priority Journals  
 EM 199701  
 ED Entered STN: 28 Jan 1997  
 Last Updated on STN: 28 Jan 1997  
 Entered Medline: 7 Jan 1997  
 AB *Helicobacter pylori* has been reported to agglutinate erythrocytes and to bind to various other cells in a sialic acid-dependent way. The binding was inhibited by **sialyllactose** or fetuin and other sialylated glycoproteins. The specificity apparently requires bacterial growth on agar, since we found that it was lost after growth in the nutrient mixture Ham's F12. Instead, the bacteria bound with high affinity and in a sialic acid-dependent way to polyglycosylceramides of human erythrocytes, a still incompletely characterized group of complex glycolipids. Bacteria grown in F12 medium were metabolically labelled with 35S-methionine and analysed for binding to glycolipids on thin-layer chromatograms and to glycoproteins on blots after electrophoresis, with human erythrocyte glycoconjugates in focus. There was no binding to simpler gangliosides

including GM3 or sialylparagloboside, or to a mixture of brain gangliosides. In contrast, polyglycosylceramides of human erythrocyte membranes bound at a pmol level. The activity was eliminated by mild acid treatment, mild periodate oxidation or sialidase hydrolysis. Erythrocyte proteins as well as a range of reference glycoproteins did not bind except band 3, which was weakly active. However, this activity was resistant to periodate oxidation. These results indicate a second and novel sialic acid-recognizing specificity which is expressed independently of the previously described specificity.

L58 ANSWER 12 OF 16 MEDLINE on STN  
 AN 95151768 MEDLINE  
 DN PubMed ID: 7849044  
 TI Enhanced binding of **enterotoxigenic Escherichia coli** K99 to amide derivatives of the receptor ganglioside NeuGc-GM3.  
 AU Lanne B; Uggl a L; Stenhammar G; Karlsson K A  
 CS Department of Medical Biochemistry, Goteborg University, Sweden.  
 SO Biochemistry, (1995 Feb 14) Vol. 34, No. 6, pp. 1845-50.  
 Journal code: 0370623. ISSN: 0006-2960.  
 CY United States  
 DT Journal; Article; (JOURNAL ARTICLE)  
 (RESEARCH SUPPORT, NON-U.S. GOV'T)  
 LA English  
 FS Priority Journals  
 EM 199503  
 ED Entered STN: 22 Mar 1995  
 Last Updated on STN: 22 Mar 1995  
 Entered Medline: 13 Mar 1995  
 AB A natural receptor in pig small intestine [Teneberg, S., Willemsen, P., de Graaf, F. K., & Karlsson, K.-A. (1990) FEBS Lett. 263, 10-14] for the **enterotoxigenic** bacteria **Escherichia coli** K99  
 is the ganglioside NeuGc-GM3 (NeuGc alpha 3Gal beta 4Glc beta Cer) [e.g., H. Smit, W. Gaasstra, J. P. Kamerling, J. F. G. Vliegenthart, & F. K. de Graaf (1984) Infect. Immun. 46, 578-584]. Chemical modifications of the carboxyl group of this ganglioside were performed, giving five different amides, the methyl ester, and the primary alcohol. The products were purified, and their structures were investigated by negative FAB mass spectrometry. Binding of **E. coli** K99 was tested by incubating 35S-labeled bacteria with derivatized compounds separated on thin-layer chromatograms. Modification of the carboxyl group to a primary amide strengthened the binding at least 5-fold, as estimated from autoradiography of dilutions on thin-layer plates. Some strengthening of the binding was also obtained with the methylamide as well as with the carboxyl group reduced to the alcohol. The ethylamide bound equally well as the underivatized NeuGc-GM3. Amide substituents as large as propyl amide and benzyl amide were still recognized by the bacteria, although they bound weaker. The methyl ester was not stable in the chromatogram-binding assay with silica gel and water present, and it reverted to the acid.

L58 ANSWER 13 OF 16 MEDLINE on STN  
 AN 95104315 MEDLINE  
 DN PubMed ID: 7528675  
 TI Alpha 2,3-sialyl and alpha 1,3-fucosyltransferase-dependent synthesis of sialyl Lewis x, an essential oligosaccharide present on L-selectin counterreceptors, in cultured endothelial cells.  
 AU Majuri M L; Pinola M; Niemela R; Tiisala S; Natunen J; Renkonen O; Renkonen R  
 CS Department of Bacteriology, University of Helsinki, Finland.

SO European journal of immunology, (1994 Dec) Vol. 24, No. 12, pp. 3205-10.  
 Journal code: 1273201. ISSN: 0014-2980.  
 CY GERMANY: Germany, Federal Republic of  
 DT (IN VITRO)  
 Journal; Article; (JOURNAL ARTICLE)  
 (RESEARCH SUPPORT, NON-U.S. GOV'T)  
 LA English  
 FS Priority Journals  
 EM 199501  
 ED Entered STN: 15 Feb 1995  
 Last Updated on STN: 6 Feb 1998  
 Entered Medline: 27 Jan 1995  
 AB Sialyl Lewis x (sLex) oligosaccharides have been shown to be present in counterreceptors for L-selectin. We and others have previously shown that high endothelial cells in lymph nodes and at sites of inflammation express sLex. Here we show that also cultured human umbilical vein endothelial cells (HUVEC) express sLex on their cell surface. This oligosaccharide is formed by sequential action of alpha 2,3-sialyl- (alpha 2,3-ST) and alpha 1,3-fucosyltransferases (alpha 1,3-FT) on N-acetyllactosamine. At least two of the several alpha 2,3-ST and four of the several alpha 1,3-FT are present in HUVEC. In functional assays both alpha 2,3-ST and alpha 1,3-FT activities were observed in HUVEC lysates with exogenous lactosamine and sialyllactosamine acceptors, leading to the generation of the sialyllactosamine and sLex sequences, respectively. TNF stimulation increased the level of mRNA expression of FT VI, and the alpha 1,3-FT activity in HUVEC. Taken together these data show that endothelial cells express sLex and that they possess mRNA as well as enzyme activities of several alpha 2,3-ST and alpha 1,3-FT necessary in the final steps of sLex synthesis. Furthermore, inflammatory cytokines such as TNF can enhance transferase activities relevant in generating putative L-selectin counterreceptors.

L58 ANSWER 14 OF 16 MEDLINE on STN  
 AN 94129215 MEDLINE  
 DN PubMed ID: 7507746  
 TI Monoclonal antibody against a lactose epitope of glycosphingolipids binds to melanoma tumour cells.  
 AU Ding K; Ekberg T; Zeuthen J; Teneberg S; Karlsson K A;  
 Rosen A  
 CS Department of Tumor Immunology, Wallenberg Laboratory, University of Lund, Sweden.  
 SO Glycoconjugate journal, (1993 Oct) Vol. 10, No. 5, pp. 395-405.  
 Journal code: 8603310. ISSN: 0282-0080.  
 CY ENGLAND: United Kingdom  
 DT Journal; Article; (JOURNAL ARTICLE)  
 (RESEARCH SUPPORT, NON-U.S. GOV'T)  
 LA English  
 FS Priority Journals  
 EM 199403  
 ED Entered STN: 18 Mar 1994  
 Last Updated on STN: 3 Feb 1997  
 Entered Medline: 8 Mar 1994  
 AB Mice were immunized with a neoglycoprotein consisting of a chemically modified carbohydrate moiety (reductively aminated 3'-sialyllactose) linked to human serum albumin. By this procedure an antibody response to the normally non-immunogenic carbohydrate structure was obtained. Hybridomas were established, and monoclonal antibodies were selected in ELISA based on their binding to the saccharide hapten, or to a lactosylceramide-mimicking neoglycolipid, lactose-bis-sulfone. One of the selected antibodies, 2H4, was of

particular interest, since it also bound to glycolipids present on melanoma cells. FACS analysis of a panel of 14 melanoma cell lines showed that the 2H4 antibody bound to the majority of these. In frozen, non-fixed sections or paraffin sections of biopsies the monoclonal antibody 2H4 stained melanoma cells, but not tumour infiltrating lymphocytes or normal skin. Detailed immunochemical analysis of 2H4, using thin layer chromatography revealed that it recognized an internal lactose epitope in several glycosphingolipids.

L58 ANSWER 15 OF 16 MEDLINE on STN  
 AN 86051591 MEDLINE  
 DN PubMed ID: 3840699  
 TI Mouse monoclonal antibodies with specificity for the melanoma-associated ganglioside **disialyllactosylceramide** (GD3) also react with the structural analogue disialylparagloboside.  
 AU Brodin T; Hellstrom I; Hellstrom K E; **Karlsson K A**; Sjogren H O; Stromberg N; Thulin J  
 SO Biochimica et biophysica acta, (1985 Dec 4) Vol. 837, No. 3, pp. 349-53.  
 Journal code: 0217513. ISSN: 0006-3002.  
 CY Netherlands  
 DT Journal; Article; (JOURNAL ARTICLE)  
 (RESEARCH SUPPORT, NON-U.S. GOV'T)  
 LA English  
 FS Priority Journals  
 EM 198601  
 ED Entered STN: 21 Mar 1990  
 Last Updated on STN: 21 Mar 1990  
 Entered Medline: 3 Jan 1986  
 AB A mouse monoclonal IgM antibody, 4.2, has previously been shown to bind preferentially to the surface of human malignant melanoma cells and to have specificity for the GD3 ganglioside (NeuAc alpha 2----8NeuAc alpha 2----3Gal beta 1----4GlcCer). Using overlay of antibodies on thin-layer chromatograms with glycolipids of various sources, it was shown that antibody 4.2, a further IgM and two IgG3 mouse monoclonal antibodies, selected on the basis of reactivity with GD3, also bound with similar strength to the structural analogue NeuAc alpha 2----8NeuAc alpha 2----3Gal beta 1----4GlcNac beta 1----3Gal beta 1----4GlcCer or disialylparagloboside. The SK-MEL 28 melanoma cell line used for immunization was shown to contain a large amount of GD3 but to lack disialylparagloboside. The demonstrated cross-reactivity may be of importance when considering the use of these antibodies for biochemical and medical purposes.

L58 ANSWER 16 OF 16 MEDLINE on STN  
 AN 80006650 MEDLINE  
 DN PubMed ID: 479198  
 TI Structural characterization of lactotetraosylceramide, a novel glycosphingolipid isolated from human meconium.  
 AU **Karlsson K A**; Larson G  
 SO The Journal of biological chemistry, (1979 Sep 25) Vol. 254, No. 18, pp. 9311-6.  
 Journal code: 2985121R. ISSN: 0021-9258.  
 CY United States  
 DT Journal; Article; (JOURNAL ARTICLE)  
 LA English  
 FS Priority Journals  
 EM 197911  
 ED Entered STN: 15 Mar 1990  
 Last Updated on STN: 15 Mar 1990  
 Entered Medline: 21 Nov 1979

AB In the course of work on a systematic structural mapping of nonacid glycosphingolipids of human meconia, special attention was given to a major component preliminarily identified as an isomer of **neolactotetraosylceramide** (paragloboside). This component was isolated in its pure form from meconium of a blood group O individual and subjected to detailed structural analyses, using mass spectrometry and proton NMR spectroscopy on intact permethylated and permethylated-reduced (LiAlH<sub>4</sub>) derivatives, and gas liquid chromatography on degradational products of native, permethylated, and permethylated-reduced derivatives. The isolated compound was conclusively shown to have the structure Galp beta 1 yields 3GlcNAcp beta 1 yields 3Galp beta 1 yields 4GlcP beta 1 yields 1Cer, and is thus identified as lactotetraosylceramide. The major fatty acids were 2-hydroxy fatty acids with 16 and 20 to 24 carbon atoms, and the bases were sphingosine and phytosphingosine. This glycolipid, although not isolated and structurally characterized before, has long been thought of as a precursor substance of the Lewis active glycolipids and of ABH-active glycolipids with a type 1 saccharide chain.

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L80 ANSWER 1 OF 4 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN  
AN 2005:275685 BIOSIS  
DN PREV200510055255  
TI Recombinant probiotics for treatment and prevention of enterotoxigenic Escherichia coli diarrhea.  
AU Paton, Adrienne W.; Jennings, Michael P.; Morona, Renato; Wang, Hui; Focareta, Antonio; Roddam, Louise F.; Paton, James C. [Reprint Author]  
CS Univ Adelaide, Sch Mol and Biomed Sci, Adelaide, SA 5005, Australia  
james.paton@adelaide.edu.au  
SO Gastroenterology, (MAY 2005) Vol. 128, No. 5, pp. 1219-1228.  
CODEN: GASTAB. ISSN: 0016-5085.  
DT Article  
LA English  
ED Entered STN: 21 Jul 2005  
Last Updated on STN: 21 Jul 2005  
AB Background & Aims: We have developed a therapeutic strategy for gastrointestinal infections that is based on molecular mimicry of host receptors for bacterial toxins on the surface of harmless gut bacteria. The aim of this study was to apply this to the development of a recombinant probiotic for treatment and prevention of diarrheal disease caused by enterotoxigenic Escherichia coli strains that produce heat-labile enterotoxin. Methods: This was achieved by expressing glycosyltransferase genes from *Neisseria meningitidis* or *Campylobacter jejuni* in a harmless Escherichia coli strain (CWG:308), resulting in the production of a chimeric lipopolysaccharide capable of binding heat-labile

enterotoxin with high avidity. Results: The strongest heat-labile enterotoxin binding was achieved with a construct (CWG308:pLNT) that expresses a mimic of lacto-N-neotetraose, which neutralized  $\geq 93.8\%$  of the heat-labile enterotoxin activity in culture lysates of diverse enterotoxigenic *Escherichia coli* strains of both human and porcine origin. When tested with purified heat-labile enterotoxin, it was capable of adsorbing approximately 5% of its own weight of toxin. Weaker toxin neutralization was achieved with a construct that mimicked the ganglioside GM2. Preabsorption with, or coadministration of, CWG308:pLNT also resulted in significant in vivo protection from heat-labile enterotoxin-induced fluid secretion in rabbit ligated ileal loops. Conclusions: Toxin-binding probiotics such as those described here have considerable potential for prophylaxis and treatment of enterotoxigenic *Escherichia coli*-induced travelers' diarrhea.

CC Pathology - Therapy 12512  
 Digestive system - Physiology and biochemistry 14004  
**Digestive system - Pathology 14006**  
 Pharmacology - General 22002  
 Physiology and biochemistry of bacteria 31000  
 Medical and clinical microbiology - General and methods 36001

IT Major Concepts  
 Pharmacology; Infection; Digestive System (Ingestion and Assimilation)

IT Diseases  
 diarrhea: digestive system disease, symptom  
 Diarrhea (MeSH)

IT Diseases  
 gastrointestinal infection: digestive system disease, infectious disease

IT Chemicals & Biochemicals  
 enterotoxin; lacto-N-neotetraose

ORGN Classifier  
 Aerobic Helical or Vibrioid Gram-Negatives 06210  
 Super Taxa  
 Eubacteria; Bacteria; Microorganisms  
 Organism Name  
*Campylobacter jejuni* (species)  
 Taxa Notes  
 Bacteria, Eubacteria, Microorganism

ORGN Classifier  
 Enterobacteriaceae 06702  
 Super Taxa  
 Facultatively Anaerobic Gram-Negative Rods; Eubacteria; Bacteria;  
 Microorganisms  
 Organism Name  
*Escherichia coli* (species): strain-CWG308  
 Taxa Notes  
 Bacteria, Eubacteria, Microorganism

ORGN Classifier  
 Leporidae 86040  
 Super Taxa  
 Lagomorpha; Mammalia; Vertebrata; Chordata; Animalia  
 Organism Name  
 rabbit (common)  
 Taxa Notes  
 Animals, Chordates, Lagomorphs, Mammals, Nonhuman Vertebrates, Nonhuman Mammals, Vertebrate

ORGN Classifier  
 Neisseriaceae 06507  
 Super Taxa  
 Gram-Negative Aerobic Rods and Cocci; Eubacteria; Bacteria;

Microorganisms  
 Organism Name  
*Neisseria meningitidis* (species)  
 Taxa Notes  
 Bacteria, Eubacteria, Microorganism  
 RN 13007-32-4 (lacto-N-neotetraose)

L80 ANSWER 2 OF 4 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN  
 AN 2001:181018 BIOSIS  
 DN PREV200100181018  
 TI Nutritional formulations containing lacto-N-neotetraose.  
 AU Prieto, Pedro A. [Inventor]; Kroening, Terry A. [Inventor, Reprint author]  
 CS Gahanna, OH, USA  
 ASSIGNEE: Abbott Laboratories  
 PI US 6083934 20000704  
 SO Official Gazette of the United States Patent and Trademark Office Patents,  
 (July 4, 2000) Vol. 1236, No. 1. e-file.  
 CODEN: OGUPE7. ISSN: 0098-1133.  
 DT Patent  
 LA English  
 ED Entered STN: 11 Apr 2001  
 Last Updated on STN: 18 Feb 2002  
 AB A nutritional formulation containing an effective amount of  
 Lacto-N-neotetraose to simulate the growth and/or metabolic activity of  
 Bifidobacterium is provided. A process of inhibiting bacterial infections  
 caused by *Bacteroides*, *Clostridium*, and *E. coli*  
 including the step of feeding the nutritional composition to a subject is  
 also provided.  
 NCL 514061000  
 CC General biology - Miscellaneous 00532  
 IT Major Concepts  
 Foods; Pediatrics (Human Medicine, Medical Sciences); Nutrition;  
 Pharmacology  
 IT Diseases  
 bacterial infection: bacterial disease  
 Bacterial Infections (MeSH)  
 IT Chemicals & Biochemicals  
 lacto-N-neotetraose: feeding, nutrient, nutritional formulations  
 ORGN Classifier  
 Irregular Nonsporing Gram-Positive Rods 08890  
 Super Taxa  
 Actinomycetes and Related Organisms; Eubacteria; Bacteria;  
 Microorganisms  
 Organism Name  
 Bifidobacterium: growth activity stimulation, metabolic activity  
 stimulation  
 Taxa Notes  
 Bacteria, Eubacteria, Microorganisms  
 RN 13007-32-4 (lacto-N-neotetraose)

L80 ANSWER 3 OF 4 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN  
 AN 1997:408381 BIOSIS  
 DN PREV199799714584  
 TI Immobilization of reducing sugars as toxin binding agents.  
 AU Nilsson, U. J.; Heerze, L. D.; Liu, Y.-C.; Armstrong, G. D.; Palcic, M.  
 M.; Hindsgaul, O. [Reprint author]  
 CS Dep. Chem., Univ. Alberta, Edmonton, AB T6G 2G2, Canada  
 SO Bioconjugate Chemistry, (1997) Vol. 8, No. 4, pp. 466-471.  
 CODEN: BCCHE. ISSN: 1043-1802.  
 DT Article

LA English  
 ED Entered STN: 24 Sep 1997  
 Last Updated on STN: 21 Nov 1997  
 AB A simple and economical procedure for the attachment of reducing sugars to aminated solid supports has been developed. Reaction of the amino groups on the solid support with p-nitrophenyl chloroformate, followed by 1,6-hexanediamine, yields a chain-extended amine to which reducing sugars can be attached while remaining accessible to macromolecules. Immobilization of the reducing sugars involves a simple incubation followed by trapping of the resulting glycosylamine with acetic anhydride and recovery of the unreacted sugar by filtration. This technique was used to immobilize lactose and sialyllactose onto silylaminated Chromosorb P, producing solid supports that effectively neutralized the activity of cholera toxin from *Vibrio cholerae* and heat-labile enterotoxin of enterotoxigenic *Escherichia coli*. The general applicability of such solid supports for toxin neutralization was further demonstrated by immobilization of the enzymatically synthesized alpha-Gal(1-3)beta-Gal(1-4)Glc trisaccharide, which produced a support that efficiently neutralized toxin A of *Clostridium difficile*. The results from this study suggest that these solid supports have the potential to serve as inexpensive therapeutics for bacterial toxin-mediated diarrheal diseases.  
 CC Biochemistry studies - General 10060  
 Biochemistry studies - Carbohydrates 10068  
 Digestive system - Pathology 14006  
 Toxicology - General and methods 22501  
 Physiology and biochemistry of bacteria 31000  
 Medical and clinical microbiology - Bacteriology 36002  
 IT Major Concepts  
 Biochemistry and Molecular Biophysics; Digestive System (Ingestion and Assimilation); Infection; Physiology; Toxicology  
 IT Chemicals & Biochemicals  
 P-NITROPHENYL CHLOROFORMATE; 1,6-HEXANEDIAMINE; SIALYLLACTOSE; LACTOSE  
 IT Miscellaneous Descriptors  
 BACTERIAL TOXINS; CHOLERA TOXIN; DIGESTIVE SYSTEM DISEASE; HOST; IMMOBILIZATION; LACTOSE; P-NITROPHENYL CHLOROFORMATE; PATHOGEN; REDUCING SUGAR; SIALYLLACTOSE; THERAPY PLANNING; TOXICITY; TOXICOLOGY; TOXIN A; TOXIN-MEDIATED DIARRHEA; 1,6-HEXANEDIAMINE  
 ORGN Classifier  
 Animalia 33000  
 Super Taxa  
 Animalia  
 Organism Name  
 animal  
 Animalia  
 Taxa Notes  
 Animals  
 ORGN Classifier  
 Endospore-forming Gram-Positives 07810  
 Super Taxa  
 Eubacteria; Bacteria; Microorganisms  
 Organism Name  
 endospore-forming gram-positive rods and cocci  
 Clostridium difficile  
 Taxa Notes  
 Bacteria, Eubacteria, Microorganisms  
 ORGN Classifier  
 Enterobacteriaceae 06702  
 Super Taxa  
 Facultatively Anaerobic Gram-Negative Rods; Eubacteria; Bacteria; Microorganisms

Organism Name  
 Escherichia coli  
 Taxa Notes  
 Bacteria, Eubacteria, Microorganisms  
 ORGN Classifier  
 Vibrionaceae 06704  
 Super Taxa  
 Facultatively Anaerobic Gram-Negative Rods; Eubacteria; Bacteria;  
 Microorganisms  
 Organism Name  
 Vibrio cholerae  
 Taxa Notes  
 Bacteria, Eubacteria, Microorganisms  
 RN 7693-46-1 (P-NITROPHENYL CHLOROFORMATE)  
 124-09-4 (1,6-HEXANEDIAMINE)  
 3001-89-6Q (SIALYLLACTOSE)  
 35890-38-1Q (SIALYLLACTOSE)  
 63-42-3 (LACTOSE)  
 11040-27-0Q (SIALYLLACTOSE)

L80 ANSWER 4 OF 4 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN  
 AN 1995:300179 BIOSIS  
 DN PREV199598314479  
 TI Inhibition of cholera toxin by human milk fractions and sialyllactose.  
 AU Idota, Tadashi; Kawakami, Hiroshi; Murakami, Yuji; Sugawara, Makihiro  
 CS Technical Res. Inst., Snow Brand Milk Products Co. Ltd., 1-1-2 Minamidai,  
 Kawagoe, Saitama 350-11, Japan  
 SO Bioscience Biotechnology and Biochemistry, (1995) Vol. 59, No. 3, pp.  
 417-419.  
 ISSN: 0916-8451.  
 DT Article  
 LA English  
 ED Entered STN: 11 Jul 1995  
 Last Updated on STN: 11 Jul 1995  
 AB The effects of human milk fractions on cholera toxin B subunit binding to  
 monosialoganglioside 1 (G-M1) were investigated. Human milk, human  
 defatted milk, whey, and a low-molecular-weight fraction of human milk  
 inhibited the binding, but casein did not inhibit it. The inhibitory  
 activity of whey from bovine-milk-based infant formula was less than that  
 of whey from human milk. Differences in composition between human and  
 bovine whey seemed to influence the extent of the inhibitory activity.  
 Sialylated oligosaccharides were considered to be the possible components  
 that inhibited cholera toxin. The effects of sialyllactose, a predominant  
 sialylated component of human milk, on cholera toxin-induced  
 diarrhea were investigated by the rabbit intestinal loop method.  
 Sialyllactose inhibited the cholera toxin inducing fluid accumulation,  
 although neither sialic acid nor lactose had an effect on it. The results  
 suggest that sialyllactose is responsible for the inhibitory activity of  
 milk on cholera toxin.  
 CC Comparative biochemistry 10010  
 Biochemistry methods - General 10050  
 Biochemistry methods - Proteins, peptides and amino acids 10054  
 Biochemistry methods - Carbohydrates 10058  
 Biochemistry studies - General 10060  
 Biochemistry studies - Proteins, peptides and amino acids 10064  
 Biochemistry studies - Lipids 10066  
 Biochemistry studies - Carbohydrates 10068  
 Biophysics - Methods and techniques 10504  
 Biophysics - Molecular properties and macromolecules 10506  
 Physiology - General 12002

Physiology - Comparative 12003  
 Pathology - General 12502  
 Pathology - Comparative 12503  
 Nutrition - General studies, nutritional status and methods 13202  
 Nutrition - General dietary studies 13214  
 Nutrition - Proteins, peptides and amino acids 13224  
 Digestive system - Physiology and biochemistry 14004  
     Digestive system - Pathology 14006  
 Reproductive system - Physiology and biochemistry 16504  
 Toxicology - General and methods 22501  
 Toxicology - Antidotes and prevention 22505  
 Physiology and biochemistry of bacteria 31000  
 Medical and clinical microbiology - General and methods 36001  
 Medical and clinical microbiology - Bacteriology 36002  
 IT Major Concepts  
     Biochemistry and Molecular Biophysics; Digestive System (Ingestion and Assimilation); Gastroenterology (Human Medicine, Medical Sciences); Infection; Methods and Techniques; Nutrition; Pathology; Physiology; Reproductive System (Reproduction); Toxicology  
 IT Chemicals & Biochemicals  
     SIALYLLACTOSE  
 IT Miscellaneous Descriptors  
     B SUBUNIT BINDING; BACTERIAL INFECTION; CASEIN; DIARRHEA; INHIBITORY ACTIVITY; LOW-MOLECULAR-WEIGHT FRACTION; RABBIT INTESTINAL LOOP METHOD; SIALYLATED OLIGOSACCHARIDES; WHEY  
 ORGN Classifier  
     Hominidae 86215  
 Super Taxa  
     Primates; Mammalia; Vertebrata; Chordata; Animalia  
 Organism Name  
     Hominidae  
 Taxa Notes  
     Animals, Chordates, Humans, Mammals, Primates, Vertebrates  
 ORGN Classifier  
     Leporidae 86040  
 Super Taxa  
     Lagomorpha; Mammalia; Vertebrata; Chordata; Animalia  
 Organism Name  
     Leporidae  
 Taxa Notes  
     Animals, Chordates, Lagomorphs, Mammals, Nonhuman Vertebrates, Nonhuman Mammals, Vertebrates  
 ORGN Classifier  
     Vibrionaceae 06704  
 Super Taxa  
     Facultatively Anaerobic Gram-Negative Rods; Eubacteria; Bacteria;  
     Microorganisms  
 Organism Name  
     Vibrio cholerae  
 Taxa Notes  
     Bacteria, Eubacteria, Microorganisms  
 RN 3001-89-60 (SIALYLLACTOSE)  
     35890-38-10 (SIALYLLACTOSE)  
     11040-27-0 (SIALYLLACTOSE)

=> => fil embase  
 FILE 'EMBASE' ENTERED AT 13:06:00 ON 07 JAN 2008  
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FILE COVERS 1974 TO 7 Jan 2008 (20080107/ED)

EMBASE is now updated daily. SDI frequency remains weekly (default) and biweekly.

This file contains CAS Registry Numbers for easy and accurate substance identification.

Beginning January 2008, Elsevier will no longer provide EMTREE codes as part of the EMTREE thesaurus in EMBASE. Please update your current-awareness alerts (SDIs) if they contain EMTREE codes.

For further assistance, please contact your local helpdesk.

=> d all

L82 ANSWER 1 OF 1 EMBASE COPYRIGHT (c) 2008 Elsevier B.V. All rights reserved on STN  
AN 2002044256 EMBASE  
TI Human milk oligosaccharides: A novel method provides insight into human genetics.  
AU Erney R.; Hilty M.; Pickering L.; Ruiz-Palacios G.; Prieto P.  
CS P. Prieto, Abbott Laboratories, Columbus, OH 43215, United States  
SO Advances in Experimental Medicine and Biology, (2001) Vol. 501, pp. 285-297.  
Refs: 12  
ISSN: 0065-2598 CODEN: AEMBAP  
CY United States  
DT Journal; Conference Article; (Conference paper)  
FS 022 Human Genetics  
025 Hematology  
029 Clinical and Experimental Biochemistry  
LA English  
SL English  
ED Entered STN: 14 Feb 2002  
Last Updated on STN: 14 Feb 2002  
AB Human milk is a unique reservoir of oligosaccharides. The presence of many of these oligosaccharides is determined genetically and is related to the Lewis blood group and secretor antigen status of each donor. A method to quantitate neutral human milk oligosaccharides was developed. Sample preparation was based on a single centrifugation-filtration step that yields oligosaccharide extracts. These extracts first were fractionated to remove a significant portion of their lactose content and were analyzed using high-pH anion-exchange chromatography. Oligosaccharide profiles from 386 milk samples obtained in this fashion generated quantitative information on lactose, the neutral cores lacto-N-tetraose (LNT) and lacto-N-neotetraose (LNneoT), and the key fucosylated oligosaccharides. Additionally, the profiles provided genetic footprints of the Lewis and secretor status of the donors. Furthermore, unusual profiles that could not have been predicted from known genotypes were found. For this reason, milk glycoproteins were studied using carbohydrate-binding probes. Results confirm that oligosaccharides are an accurate predictor of the Lewis blood group status of the donor, and that glycosyltransferases have exquisite specificities. The data obtained in this study corroborate that Lewis-related antigens are tissue specific. This attribute of immunodominant carbohydrate sequences has significant implications for epidemiological studies of breast-fed infants.  
CT Medical Descriptors:  
accuracy

anion exchange chromatography  
 antigenicity  
 biochemical composition  
 blood group Lewis system  
 \*breast milk  
 carbohydrate analysis  
 centrifugation  
 conference paper  
 extract  
 filtration  
 fractionation  
 genotype  
 human  
 human genetics  
 prediction  
 priority journal  
 protein determination  
 technique

## CT Drug Descriptors:

antigen: EC, endogenous compound  
 carbohydrate: EC, endogenous compound  
 glycoprotein: EC, endogenous compound  
 glycosyltransferase: EC, endogenous compound  
 lacto n neotetraose: EC, endogenous compound  
 lacto n tetraose: EC, endogenous compound  
 lactose: EC, endogenous compound  
 \*oligosaccharide: EC, endogenous compound  
 unclassified drug

RN (glycosyltransferase) 9033-07-2; (lacto n neotetraose) 13007-32-4  
 ; (lactose) 10039-26-6, 16984-38-6, 63-42-3, 64044-51-5

=> => d his

(FILE 'HOME' ENTERED AT 12:30:48 ON 07 JAN 2008)  
 SET COST OFF

FILE 'REGISTRY' ENTERED AT 12:31:01 ON 07 JAN 2008

L1 1 S 35890-39-2  
 L2 1 S 35890-38-1  
 L3 1 S 13007-32-4

FILE 'MEDLINE' ENTERED AT 12:31:35 ON 07 JAN 2008

L4 0 S L1  
 L5 277 S ?SIALYLLACTOS? OR ?SIALYLL LACTOS? OR ?SIALYLACTOS? OR ?SIALY  
 L6 30 S ACETYL(S)NEURAMIN?(S)LACTOS?  
 L7 179 S ACETYLNEURAMIN?(S)LACTOS?  
 L8 1 S NAN LACTOS?  
 L9 425 S ?NEURAMINYLLACTOS? OR ?NEURAMIN? ?LACTOS?  
 L10 1 S NAN LACTOS?  
 L11 363 S ?NEURAMIN? ?LACTOS?  
 L12 20 S LNNT  
 L13 73 S NEOLACTOTETRAOS?  
 L14 162 S LACTO (S) (NEOTETRAOS? OR NEO TETRAOS?)  
 L15 1014 S L5-L14  
 L16 6 S L15 AND (?DIARRH? OR ?DYSENTER? OR ?COLIC?)  
 E DIARRHEA/CT  
 E E3+ALL  
 L17 35903 S E5+NT  
 E E9+ALL

L18 1224 S E6  
     E DIARRHEA/CT  
     E E4+ALL  
     E E2+ALL  
 L19 367 S E41  
     E DIARRHEA/CT  
     E E5+ALL  
     E E9+ALL  
 L20 1577 S E9+NT  
     E DYSENTERY/CT  
     E E3+ALL  
 L21 9964 S E9+NT  
     E E8+ALL  
 L22 116171 S E4+NT  
     E COLIC/CT  
     E E3+ALL  
 L23 2978 S E36  
 L24 1 S L15 AND L17-L23  
 L25 6 S L16, L24  
 L26 58 S L15 AND (ESCHERICHIA OR "E") ()COLI  
     E ESCHERICHIA/CT  
     E E4+ALL  
 L27 43 S L15 AND E11+NT  
 L28 9 S L15 AND E32+NT  
 L29 44 S L15 AND E10+NT  
 L30 59 S L26-L29  
     E ENTEROTOXIN/CT  
     E E5+ALL  
 L31 19 S L15 AND E4+NT  
 L32 71 S L25, L30, L31  
 L33 12 S L15 AND ?ENTEROTOX?  
 L34 71 S L32, L33  
     E GASTROINTESTINAL DISEASE/CT  
     E E4+ALL  
 L35 30 S L15 AND E3+NT  
 L36 98 S L34, L35  
 L37 98 S L36 AND L1-L36  
 L38 78 S L37 AND PY<=2002  
 L39 20 S L37 NOT L38  
 L40 1 S L39 AND 2005246876/AN  
 L41 2 S L38 AND (79216779 OR 95252586)/AN  
 L42 7 S L40, L41, L25 AND L1-L41  
 L43 3 S L42 AND ?CERAMID?  
 L44 7 S L42, L43  
     E ANGSTROM/AU  
 L45 59 S E5, E6  
     E TENEBERG/AU  
 L46 60 S E4, E5  
     E SAARINEN/AU  
 L47 81 S E3, E14-E16, E18  
     E SATOMAA/AU  
 L48 3 S E6  
     E ROCHE/AU  
 L49 15 S E3  
     E ROCHE N/AU  
 L50 116 S E3-E6, E13  
     E NATUNEN/AU  
 L51 15 S E4, E5  
     E MILLER PODRAZA/AU  
 L52 46 S E4, E5

E MILLER H/AU  
E PODRAZA/AU  
L53 2 S E9  
E KARLSSON/AU  
E KARLSSON K/AU  
L54 391 S E3, E4  
L55 18 S E16-E18  
E ABUL/AU  
E ABUL M/AU  
L56 12 S E7, E8  
E ABULMILH/AU  
L57 16 S L15 AND L45-L56  
L58 16 S L57 AND L1-L57

FILE 'MEDLINE' ENTERED AT 12:57:53 ON 07 JAN 2008

FILE 'BIOSIS' ENTERED AT 12:58:22 ON 07 JAN 2008

FILE 'MEDLINE' ENTERED AT 12:58:33 ON 07 JAN 2008

L59 99 S L2 OR L3  
L60 1 S L59 AND (?DIARRH? OR ?DYSENTER? OR ?COLIC?)  
L61 0 S L59 AND L17-L23  
L62 4 S L59 AND (ESCHERICHIA OR "E")()COLI  
L63 4 S L59 AND ESCHERICHIA+NT/CT  
L64 1 S L59 AND ENTEROTOXINS+NT/CT  
L65 3 S L59 AND GASTROINTESTINAL DISEASES+NT/CT  
L66 8 S L60-L65  
L67 7 S L66 NOT L44, L58

FILE 'BIOSIS' ENTERED AT 13:00:56 ON 07 JAN 2008

L68 13 S L1  
L69 170 S L2  
L70 48 S L3  
L71 216 S L68-L70  
L72 13 S L71 AND (ESCHERICHIA OR "E")()COLI  
L73 3 S L71 AND (?DIARRH? OR ?DYSENTER? OR ?COLIC?)  
L74 2 S L71 AND ?ENTEROTOX?  
L75 14 S L72-L74  
L76 2 S L75 AND (2001:181018 OR 1995:300179)/AN  
L77 9 S 14006/CC AND L71  
L78 6 S L77 NOT L75  
L79 3 S L77 NOT L78  
L80 4 S L76, L79

FILE 'BIOSIS' ENTERED AT 13:04:07 ON 07 JAN 2008

FILE 'EMBASE' ENTERED AT 13:04:15 ON 07 JAN 2008  
L81 25 S L1-L3  
L82 1 S L81 AND 2002044256/AN

FILE 'EMBASE' ENTERED AT 13:06:00 ON 07 JAN 2008